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MECHANISMS UNDERLYING CARDIAC DYSPNEA

Part II

The Nervous Element and the Occasional Factors

It is characteristic of cardiac dyspnea that it has occasional, especially nocturnal, incidence and that the volume of air actually moved per minute is excessive in relation to the gaseous metabolism. The explanation of these facts rests not on the organic changes and their purely mechanical results, but to a large extent on the disturbance in the nervous control of respiration. Respiration is so coordinated as to maintain a very constant arterial oxygen tension and acidity. When fixed acid accumulates or alkali is lost from the body, the respiration is increased by reflexes from nerve endings (chiefly in the carotid body and sinus), sensitive to the change in blood composition. Thus alveolar CO_2 tension and the CO_2 in the blood are reduced and normal blood alkalinity maintained. When respiration is impaired and CO_2 tension rises in the blood and alveolar air, the acidity can be regulated by renal acid excretion, but the fall in O_2 tension can only be corrected by putting the patient in an atmosphere rich in oxygen, which allows alveolar and hence arterial O_2 tension to rise even though lung ventilation remains low and CO_2 tension high. These reflex changes in respiration, evoked by changes in blood O_2 and acid content, are the paramount controls for ventilation rate; it is they which determine the hourly and daily volume of lung ventilation. From time to time the activity of the respiratory center may be dominated by other reflexes—those concerned with phonation, eating, defecation, coughing, sneezing, etc.; those evoked by cold, pain, and powerful emotion or by muscular activity or by distention of the chambers of the heart. These reflexes cause respiration to increase on effort even before blood from the active muscles affects the regions sensitive to chemical change. Such non-chemical reflexes serve to prevent the respiratory apparatus from being "caught napping" when there is a sudden increase in metabolic activity and blood flow, and they also play a rôle in determining the occasions for dyspnea. Nevertheless, respiration in heart disease is not often excessive in relation to arterial CO_2 tension. On the other hand the sense of suffocation is rarely due, except in terminal stages, to arterial acidosis or anoxemia. Cardiac dyspnea can rarely be ascribed either to inadequate blood flow to vital centers, or to over-ventilation leading to alkalosis.

A. Rapid, Shallow Breathing. Distention or deflation of a lung stimulates vagal endings in the lung which have inhibitory action on the respiratory center and thus tend to cut short either inspiration or expiration. If the vagi are cut, breathing becomes slow and deep, but if the lung is inflamed, edematous or hyperemic the endings are more sensitive and respiration becomes rapid and shallow. Since 100 to 150 cm. of air is taken up by the dead space in the bronchi, trachea and pharynx and this is wasted so far as alveolar ventilation is concerned, rapid breathing is inefficient because it moves more of this useless air each minute, and if the breathing is shallow, the waste air may represent 50-70%, instead of the normal 30% of each breath. Hence the cardiac with his sensitized pulmonary reflexes will have to move more air per minute with rapid, shallow breaths, to secure normal lung ventilation. The hyperpnea of the cardiac is due to the extra air needed to maintain normal arterial CO_2 and O_2 tension, either because of rapid breathing or, at the end of the struggle, to more difficult diffusion of gas through thickened alveolar walls.

B. Evening Dyspnea. The cardiac naturally has his difficult breathing accentuated by the extra metabolic needs and blood flow occurring during exertion, digestion or fever. He also has a greater tendency to dyspnea in the evening than in the morning, even when at rest and sitting up. During the day metabolism rises and CO_2 output is greater, at the same time the respiratory center is more active and a lower arterial and alveolar CO_2 tension is maintained. This diurnal swing in blood

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CO_2 tension is less marked in cardiacs than in normals, but the total effect is to increase the ventilation needed. Meanwhile eating, drinking and afternoon rise in temperature lead to rise in venous pressure and fall in vital capacity. Hence the cardiac has to move 30-40% more air toward evening than in the morning, and can breathe less easily because of greater lung engorgement.

C. Orthopnea. Orthopnea which is common in cardiacs, also occurs in patients with marked pleural effusions or great abdominal distention, for purely thoracic breathing is made easier and more effective if the diaphragm is low, and a fall of the diaphragm does occur on sitting up. In tricuspid valve disease and when concretio cordis obstructs venous return to the right auricle, the patient rarely experiences orthopnea, which, however, is often present and is sometimes an early sign of left heart failure and rise in pulmonic venous pressure. So we must look for the cause of orthopnea in the lungs themselves. When one is standing or sitting much blood is trapped in the veins of the abdomen, lower trunk and legs, for the venous pressure there rises considerably in order to lift the blood to the level of the heart. When one lies down the pressure in the veins falls and they become smaller. The extra blood held in them is freed and may augment the effective venous pressure in the auricles. Hence cardiac output increases slightly in the recumbent posture and this implies a slight increase in the work of the heart and in pulmonary venous pressure.

In the erect position only a little of the lung is dependent, with reference to the left ventricle into which the blood from the lung must flow, but when recumbent more than two-thirds of the lung is dependent and the venous pressure in the dependent part rises (as it does in the legs when standing) so that the blood will flow up to the left ventricle. The center of mass of the lungs is 3 to 6 cm. above the left ventricle when one sits up, 3.5 to 7 cm. below it when one lies down, so that the average venous pressure in the lung must be 7 to 10 cm. of H_2O greater when recumbent than when erect, merely to provide a constant level of pressure in the left ventricle during diastole and thus maintain a constant cardiac output. The lungs consist of the spongiest tissue in the body and edema forms in them even more readily than in subcutaneous tissue, in spite of the speed of evaporation of alveolar transudate. When venous pressure is already high due to heart failure the recumbent posture lowers vital capacity (as it does even in normals) and hastens edema formation. Discomfort may be manifest at once, or only after being recumbent for several hours.

D. Nocturnal Dyspnea and Cheyne-Stokes' Breathing. Nocturnal dyspnea may be merely latent orthopnea, wakening the sleeper when lung edema becomes marked; it may be due to acute pulmonary edema. The latter usually seems to occur with an increased burden on the weakened left ventricle, or following excessive salt or water ingestion which hastens edema formation, and in some instances it is thought to be "neurogenic," that is, to arise from vasomotor nerve imbalance in the pulmonary circuit. Cough also is an aggravating factor and acute lung edema due to bronchitis is not rare in cardiacs. Finally, no turnal dyspnea is often due to the hyperpneic pha of periodic (Cheyne-Stokes') breathing. The a neic part of the cycle, so alarming to observer does not trouble the patient who is disturbed on by dyspnea due to the periods of overbreathin In younger cardiacs, even those in extremis, suc breathing is very rare, but in the older ones ma be present off and on for months or years. Thi tendency is most marked when drowsy or gorge with food, and it is regularly aggravated by mo phine, diminished by caffeine or metaphyllin, an sometimes gradually cleared by breathing 40-70 oxygen mixtures. 5% CO_2 causes steady but e cessive respiration and is not pleasant for th patient, while morphine, exaggerating the disturb ance, may yet allow the sufferer to sleep.

The height of Cheyne-Stokes' hyperpnea coin cides with the flow through the radial artery (an presumably through the regions whose nerves ar sensitive to blood composition) of blood very poo in O_2 and 10-15% richer than normal in acid ion This of course was the blood which passed throug the alveoli toward the close of the apneic perio and which took 12-20 seconds to reach the ner endings. The ventilation rate drops rapidly as th arterial blood becomes more normal, and cease 5-10 seconds before the best aerated blood reache the arteries. This blood is only normally alkalin and cannot be the inhibitory stimulus which re presses breathing for the ensuing 20-40 seconds i spite of increasing arterial acidity and anoxemia When a patient subject to this disorder is breathin regularly a cough or sign often is immediatel followed by apnea which starts the cycles going This occurs before blood aerated by the extra ac tivity of the muscles could reach the chemically sensitive nerves, and points to inhibition by a reflex from fatigued respiratory muscles as th cause of the apnea. In any cardiac breathing rap idly against the resistance of boggy lungs, bu especially in old people with poor vascular bed and rigid chest walls, or in aortics with lo diastolic pressure, the respiratory muscles suffe greatly and can only rest during a period of apnea It seems very probable that in heart disease periodic decrease or cessation of breathing is du to fatigue and anoxemia of all the muscles of re piration, but is made possible by the lag betwee aeration of the blood and its arrival in regions sen sitive to its gas content. The inhibition of breath ing, grateful to tired muscles, unfortunately lead to hyperpnea just when the arterial blood is mos anoxic and least useful to the muscles, and it als is likely to cause dyspnea which, occurring just a the patient drops off to sleep, is most distressing

Summary: Reflexes from the lungs and fro the respiratory muscles are of great importanc in determining the character of breathing. Th inefficient, rapid, shallow breathing is due to re flexes from the engorged lung; periodic breathin in cardiacs to reflexes from fatigued respirator muscles. These factors and the rise in pulmoni venous pressure on lying down are held to ac count for the frequency of evening and nocturna dyspnea.

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